REDUPLICATION,

OR DOUBLING,

OF THE CARDIAC SOUNDS.

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Although the term reduplication is not etymologically correct, yet it has the merit of being well recognised and long established There are two sounds of the heart, one or other or both of which may be doubled but not reduplicated; and so, for my own part, I prefer the phrase, Duplication of the Cardiac Sounds, as a more accurate definition of the phenomena in question, and although I still retain the title reduplication, I shall seldom find it necessary to make use of that word in these pages.

In the *Medical Times and Gazette* of January and February 1877, I published a rather lengthy paper on "Reduplication of the Cardiac Sounds," and beyond dissenting from a more or less universally adopted name, I have since found no reason to alter in any particular the views which I then expressed.

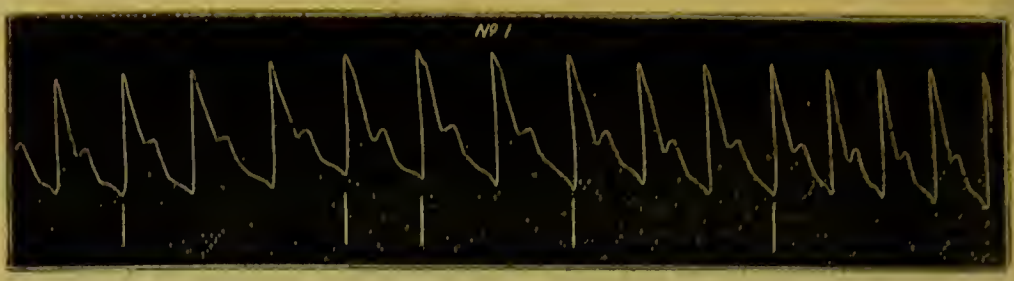
Doubling of the cardiac sounds has attracted considerable attention of late, and has given rise to great diversities of opinion as to its causation and mechanism. As there is more unanimity of

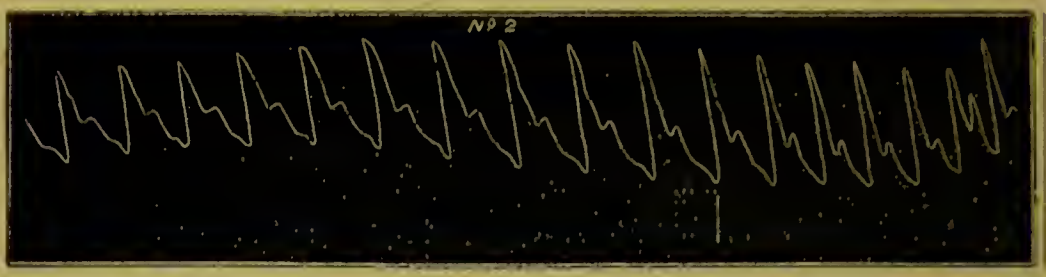
opinion regarding the causation of the second than of the first sound of the heart, so there has been less contention about the origin of a duplex second sound than of a double first. Perhaps it would be well if, before entering into any discussion on this subject, I broadly and briefly stated my own views on these matters. I believe that the normal first sound is more or less complex in its origin, and is made up of the sound of muscular contraction, the ventricular impulse against the chest walls, and the sound resulting from the simultaneous tension of the tricupsid and mitral valves. These elements of sound —namely, muscular sound, impulsion sound, and sound of valve tension — have variable participations in the amount and character of the sound, according as it is heard at the apex or over the body of the ventricles. The normal second sound is produced by the sudden and simultaneous closure with tension of the pulmonic and aortic semilunar valves at the end of ventricular systole, or in the initial stage of ventricular relaxation.

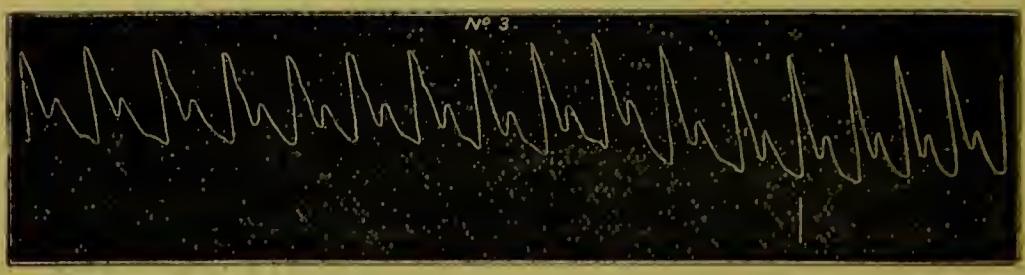
With regard to duplication of the cardiac sounds, I believe that to be due to a resolution into the component valvular elements of the sounds. I therefore hold that duplication of the first sound is due to want of synchronism in the closure and tension of the tricupsid and mitral valves, or, in other words, in the initial stage of contraction of the right and left ventricles, and I ascribe doubling of the second sound to asynhcronism at the end of ventricular contraction, and in the consecutive reaction of the pulmonary artery and aorta, with tension of their respective valves. In the case of a duplex first sound, the valvular sound of the first or of the second element may be reinforced by the sound of impulsion, and one or other or both elements may have, more or less marked, the somewhat prolonged character of the muscular sound. On account of the complexity, and consequently lengthened nature, of the first sound, a greater amount of asynchronism is required between the closure and tension of the tricupsid and mitral valves, than between the closure and tension of the pulmonic and aortic semilunar valves, to produce perceptible duplication, hence a double first sound is not so common as a double second.

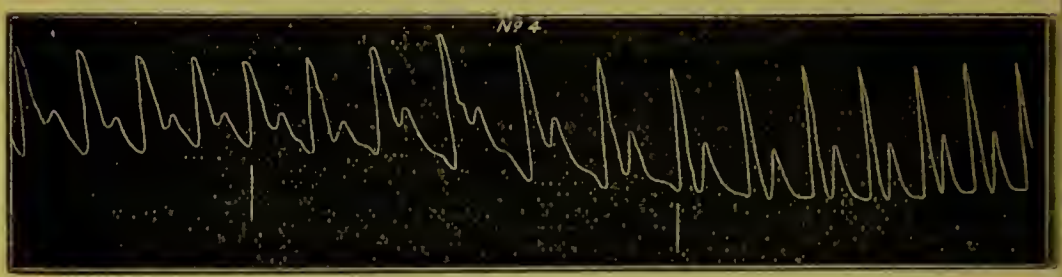
Among those who hold by the wide and comprehensive term of asynchronism, there is perhaps as little real unanimity of opinion, as there is between them and those who deny that there is any such thing as asynchronous ventricular contraction. Some, such as the late Dr. Leared, may acknowledge the possibility of asynchronism, but would not admit the valvular origin of the cardiac sounds. Others, as the late Dr. Hayden, explain a duplex second sound by asynchronous closure of the pulmonic and aortic semilunar valves, but do not allow an analogous mechanism as the cause of a double first sound. Again, there are others, who perhaps disliking to be tied down to the narrow limit of Newton's dictum, "that we are to admit no more causes of natural things than such as are both true and sufficient to explain their appearance," have devised various theories. For instance, Guttmann, in addition to his auricular theory, believes that many cases of double first sound may be due "to *non-synchronous* tension of the individual segments of the auricular ventricular valves, owing to absence of perfect uniformity in the contraction of the papillary muscles." This hypothesis—for it is unworthy of the name of theory—is, in my opinion, as absurd as the explanation some give of mitral adynamic murmurs, by supposing choreic twitching of the musculi papillares. They seem to forget, that each set of papillary muscles is connected, not with one cusp, but with all the cusps of each valve. Guttmann's explanation of a duplex second sound, by imagining asynchronous closure of the individual segments of the semilunar valves, is a physical absurdity, He has also two other theories to account for duplication of the second sound, namely, non-simultaneous closure of the pulmonary and aortic valves, and an auricular theory.

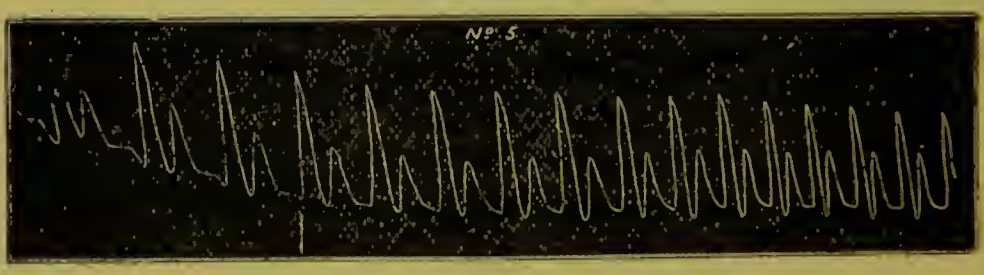
So much for the divergence of opinion among the theorists of the asynchronous origin of duplication. We shall now see that there is perhaps a greater difference, as to the causes and mechanism of asynchronism. Previous to my writings on this subject, those who held what I may briefly term the theory of asynchronous valvular tension, accounted for doubling of the first sound by supposing that excess of blood pressure in one or

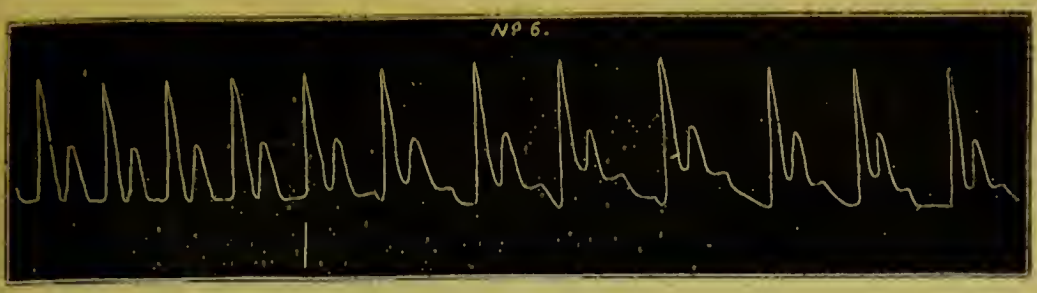

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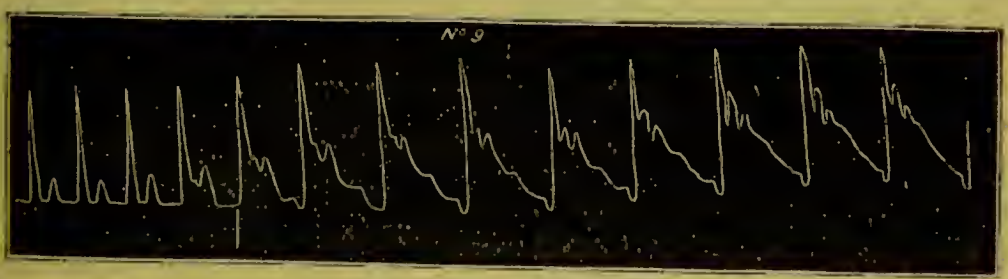

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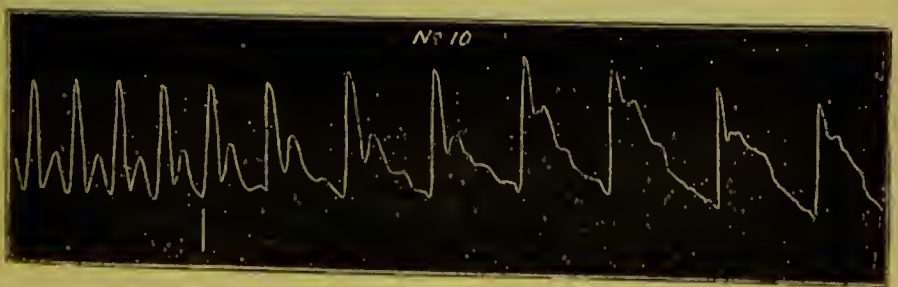

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other ventricle, retarded the closure of its auriculo-ventricular valve; and, for a duplex second sound, by supposing that excess of blood pressure in the aorta or pulmonary artery accelerated the occlusion of the respective valve. I, at least to my own mind, clearly demonstrated, that relatively greater blood supply to one or other ventricle does not retard the closure of its auriculo-ventricular valve, but more quickly overcomes the inhibitory action of the vagus, stimulates that ventricle to initiate contraction, and first apply tension to its auriculo-ventricular valve, which perhaps may be the more readily effected on account of the hyper-distension of the ventricle, and thus produces the first element of a duplex first sound. With regard to the second sound, I showed that duplication did not directly depend on high tension in one or other artery, but was owing to asynchronism at the end of ventricular contraction, and in the consecutive reaction of the pulmonary artery and aorta, with tension of their respective valves. The period of systole depends on the quantity of blood to be driven, the power and activity of the ventricle to drive it, and the resistance offered to the issuing current; and, *cæteris paribus*, the less the resistance the shorter the systole.

Physiological doubling of the first sound occurs at the end of expiration or commencement of inspiration, and of the second sound at the end of inspiration or commencement of expiration. It will be thus seen that normal duplication is dependent on the respiration, and hence might appropriately be termed respiratory doubling of the cardiac sounds, though perhaps it will be better to retain the adjective physiological, in contradistinction to pathological doubling, which we shall have to consider further on.

In my paper in the *Medical Times and Gazette*, I published some pulse tracings, taken from Dr. D. J. Jones, showing the effects of respiration on the circulation in the state of perfect health, and, as I have not since obtained any better, I reproduce them above with their interpretations.

INTERPRETATION OF THE SPHYGMOGRAPHIC TRACINGS.

No. 1 was taken during quiet respiration, but even here the effect of that process on the circulation is well marked. The first curve may be said to represent the state at the end of slight inspiration; the next three the condition in expiration; the fifth that in slight inspiration; the sixth and seventh the state in expiration; the following three that in inspiration; and the remainder with the breathing suspended at the end of the last stage.

No. 2 was taken during prolonged and full inspiration, which was at first slow, and then rapid, followed by suspension of breathing. In the inspiratory part the base line gradually descends, thus indicating the decrement in the quantity of blood in the artery between the pulsations. The height of the up-stroke greatly increases, but it quickly collapses, thus giving the short sharp shock of low tension, as shown by the approximation of the aortic notch to the base line, and the indistinctness of the tidal wave. The frequency of the pulse remains pretty unifom, or rather lessens, the distance from the upstrokes being from 6·2 to 6·5 millimetres, and the period of systole, as measured by the distance from the upstroke to the aortic notch is tolerably well sustained. When the breathing is suspended, in the state of full inspiration, the low tension is even more marked, the pulse increases in frequency so that the curves at last measure only 4·5 millimetres, and the length of the systole falls from 2 to 1·5 millimetres.

No. 3 was taken during prolonged and full inspiration, followed for the last three beats by suspension of breathing. In this case the conditions are almost similar to those of No. 2, but the diminution in the tension is even more decided.

No. 4 was taken during slow, followed by rapid and full inspiration, and then while the breath was held in the latter state. In the first place the tension is rather low, the pulse is frequent, the individual curves only measuring 5·5 millimetres, but the systole is of fair duration, as it measures 2 millimetres. In the rapid and full inspiratory part the base line descends; the up-

stroke is vigorous; the tension diminishes; the frequency of the pulse lessens, so that the curves reach 8 millimetres; and the systole is of good length. In the latter part, or that of full inspiration, the base line remains on a level; the dicrotism is great; the length of the pulse-curves gradually increases to 5·5, and that of the systole to 1·5 millimetres.

No. 5 exhibits the condition at the end of full inspiration, followed by suspension of breathing with the chest fully expanded. The base line of the first three curves quickly descends; the dicrotism is full; but the curves are long, and the systole is well sustained. In the latter part the base line remains stationary; the tension is so low that hyperdicrotism becomes very distinct; the frequency of the pulse is increased, so that at last the curve only measures 4·8 millimetres, and the period of systole is reduced to 1·5 millimetres.

No. 6 shows the state during full inspiration, followed by deep expiration. In the first part there is a good upstroke, full dicrotism, a pulse of moderate frequency, and ordinary systole. In the expiratory stage the pulse becomes very slow, so that the curves measure from 9 to 10 millimetres, and the systole is prolonged to about 2·5 millimetres. The tidal wave is fairly sustained, and as many as two undulations are noticeable after the aortic curve.

No. 7 indicates the condition during deep expiration, followed by slight inspiration. In the expiratory part the base line slightly rises, the tension increases, the frequency of the pulse lessens, and the systole is lengthened. In the slight inspiration, the tension is lowered, the frequency of the pulse increases, and the period of systole is lessened.

No. 8 was taken while the breathing was suspended in the state of deep expiration. In this instance the base line remains on the same level, and the individual curves are very uniform. The percussion wave is good, the tension fair, and four to five undulations appreciable in each curve. The pulse is infrequent; each curve measuring about 7 millimetres; and the period of systole is increased to 2·5 millimetres.

Nos. 9 and 10 represent the state at the end of full inspiration

followed by deep expiration. In the first part the tension is very low, the pulse frequent, and the systole short; but during the expiratory stage the base line rises, the tension greatly increases, the pulse becomes slower, and the systole is slightly lengthened.

Summary.

1 *During inspiration* there is an accumulation of blood in the great veins of the chest and in the pulmonary circuit, which gives a good supply to both ventricles, and hence there is then perfect synchronism in the initial stage of contraction. The aspiration of the thorax increases the capacity of the pulmonic system, thus primarily lessens the obstructive burthen of the right ventricle, and gives a freedom to the pulmonary flow throughout the whole period of inspiration, and that proportionate to the extent of expansion. As not merely the rapidity of the pulse, but also the duration of systole, greatly depend on the amount of resistance—the less the resistance, the shorter the systole,—so it follows that the right ventricular systole will occupy less time at the end, than at the beginning of inspiration, or indeed, than at any other time. It is true that there is a similar increase in the capacity of the systemic circuit, by the indirect action of lessening the quantity of blood in it; but then the assistance rendered to the left ventricle is only in proportion to that given to the right, as the quantity of blood in the systemic is to that in the pulmonic system. Accordingly we find in the tracings Nos. 2, 3, 4, and 5, that, although the tension is diminished, the frequency is not increased, but is rather lessened, and the systole is well sustained, which can be accounted for by the increased supply of blood to the left ventricle. Besides the indirect aid to the left ventricle is not rendered *pari passu* with the direct assistance afforded to the right ventricle, for we find that when the chest is fully expanded, and consequently all further succour withheld from the right ventricle, yet the tension in the systemic arteries still diminishes, the frequency of the pulse is increased, and the period of systole is lessened. (See tracings Nos. 1, 2, 3, 4, and 5.) From the

foregoing it may be easily adduced that *at the end of inspiration*, a degree of asynchronism may be established at the end of ventricular contraction, and in the consecutive reaction of the pulmonary artery and the aorta, which would give rise to a reduplicate second sound, the primary element being pulmonic.

2 *When the breathing is suspended in the state of full inspiration* the aspirating force has ceased, so there is no further aid given to the right ventricle. The thorax now contains its maximum quantity of blood, and consequently the systemic system its minimum, which latter condition is well exhibited in tracing No. 5. Any abstraction, in the course of the circulation, from the repletion of the intra-thoracic vascular cavity is quickly refilled from the systemic veins by the atmospheric pressure. This good and equal supply of blood hastens the cardiac action (Nos. 1, 2, 3, 4, and 5), and equally stimulates each ventricle to begin contraction. Now the mass of blood in the lungs becomes an obstacle to the right ventricle, which prolongs its systole, while at the same time the low tension in the systemic vessels shortens the left ventricular systole, so that a balance is again struck, with restoration of synchronism, and abolition of the reduplication of the second sound which existed at the end of inspiration.

3 *At the commencement of expiration following on full inspiration* the contraction of the chest-walls reduces primarily and mainly the aerial thoracic cavity, and, I may say, only negatively the blood vascular cavity, which, being essentially a closed circuit, cannot be altered in dimensions by the resiliency of the lungs, and therefore remains unaffected. So at the beginning of expiration the right ventricle is not at once appreciably affected, but, the aspirating force being removed, accummulation of blood at once begins in the systemic radicles, with increase of tension and prolongation of systole, as will be seen in tracings Nos. 6, 9, and 10, so that reduplication of the second sound can still be detected; and this phenomenon might continue throughout the whole expiratory period were it not that the overloading of the right ventricle and the greater obstruction in front lengthen its systole also.

4 *During expiration* both ventricles are well supplied with

blood,—in the case of the right, owing to the accumulation in the great veins; and in that of the left, owing to the amount stored up in the lungs during inspiration. Again, in both systems arterial tension is high, so synchronism is maintained both at the beginning and end of ventricular contraction. (See tracings Nos. 6, 7, 9, and 10.)

5 *At the end of expiration* the great veins of the chest have become engorged, while the surplus in the lungs has been disposed of, so the right ventricle becomes over distended, and is stimulated to initiate contraction, thus giving rise to a reduplicate first sound; but the same overloading and the increased obstruction in front prolong the right ventricular systole, hence there is no doubling of the second sound.

6 *When the breathing is suspended in the state of deep expiration*, accumulation of blood has taken place in the systemic vessels, so that they now contain their maximum quantity and their tension continues uniform; the curves are equal, and also the periods of systole, as will be seen from tracing No. 8. This cumulation reduces that in the thorax to its minimum—both that in the great veins to the right heart, and that in the lungs to the left,—so that, as far as the heart is concerned, an equable distribution of blood is maintained, and thus, all disturbing influence being removed, synchronism is restored.

7 *At the beginning of inspiration following on deep expiration* the right ventricle is the first to be over-repleted, which stimulates it to initiate contraction, and thus we again get a double first sound; but as inspiration goes on, both ventricles are equally well filled, hence there is abolition of the reduplication.

The respiratory movements exert a great influence over the circulation, even to the periphery, and give rise to a relative disturbance in the balance of the blood supply in the two sides of the heart, and in their respective arteries, sufficient to account for the phenomena in question. The primal elements of physiological doubling of both sounds are associated wlth the right side of the heart.

Pathological doubling is not merely an occasional occurrence at certain periods of respiration, but is a more or less constant

event, and, unlike normal doubling, its primal elements may be associated with either ventricle. The ventricle which is relatively the best supplied with blood, and which best retains its muscular irritability, has the inhibitory action of the vagus first overcome, first raises its intra-ventricular pressure, and tensely closes its auriculo-ventricular valve, which gives rise to the first element of a double first sound. It does not necessarily follow that the ventricle which begins will first end contraction, for the period of systole varies. The period of systole depends on the mass to be moved, the power of the ventricle to move it, and the resistance in front, and *cæteris paribus* the greater the resistance the longer the systole. Frequently the ventricle which begins contraction has proportionately the greatest amount of work to do, so its systole is protracted, and hence a double second sound is not a necessary accompaniment of a double first. Again, both ventricles may commence their systole together, but one, having in proportion to its power relatively less work to do, may end before the other; hence a double second sound. High arterial tension is a frequent source of duplication, but it is not the immediate but only the indirect agent. It is not the high tension *per se* which causes the duplication, but it is the effects which it produces in the capacities and irritability of the ventricles and its interference with the uniform distribution of blood on the one hand, and its leading to protracted ventricular systole on the other.

One or other or both venticles are sometimes converted into a species of blood reservoirs, through dilatation and failure in the muscular force, so that only part of the blood is expelled during systole. Thus the absence of duplication in some cases where we might have expected it to be present is readily explained, and in other cases the association of the primal elements with one ventricle where *á priori* we might have expected them to be connected with the other.

In reply to the objection which has been urged against the possibility of asynchronism, on account of the interlacement of the cardiac fibres and the observed consentaneousness of the ventricular action, I say that I have seen, felt, and heard

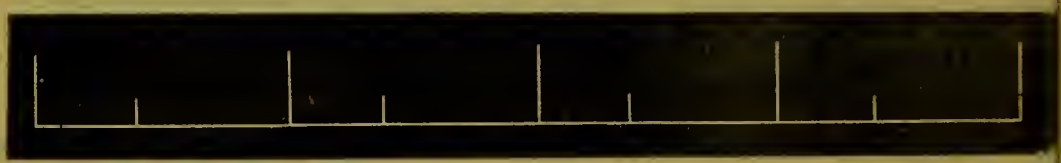
asynchronous action, and so have no difficulty in admitting the possibility of its occurrence. I do not say that such asynchronism as exists between the auricles and ventricles is possible between the two ventricles, but I do say that each side of the heart has its own proper muscular fibres, as well as those which are common to both sides; and those proper fibres form in great part the deepest layers, and so are first subjected to the stimulating influence of distension in producing contraction; and in those fibres which are common to both sides it is possible for the wave of contraction to commence, as it were, at one extremity of the fibre and be propagated to the other. Each side has more or less independently its own nerve supply, its own peristaltic action, and, notwithstanding that both sides are set to the same time, and that there is a complex interlacement of fibres, yet it is quite possible, nay, it is an experimental fact, that one side may begin or end contraction before the other.

The following is an extract from my notes of an unique case of almost complete asynchronous ventricular contraction, which came under my care in the Stanley Hospital :—

Isabella Watt, aged 31, waitress, single, admitted to the Stanley Hospital March 3, 1880, suffering from cardiac dyspnœa. She had two attacks of rheumatic fever—one when nine years of age, and the other about seven years ago. From this latter time she kept in pretty fair health until February 1879, when, owing to family sickness, she had a great deal of work to do both day and night. She then began to suffer from shortness of breath on the least exertion, and her legs were frequently swollen at night, but she continued at work up till September, when she was compelled to leave off, and was admitted to the Royal Infirmary, under the care of Dr. Glynn, October 21st, 1879. The cardiac phenomena when under Dr. Glynn may be briefly summarised as follows :— The impulse was felt in the sixth interspace, in the nipple line, where there was also felt a presystolic thrill. There were heard presystolic and systolic mitral murmurs, and two short, sharp second sounds, which at times were audible over the whole præcordium. Pulse varied from 30 to 88 per minute. She left the Infirmary about the end of November, 1879, and was admitted to the Stanley Hospital under my care, on March 3rd, 1880. The apex beat was then situated in the sixth interspace about one inch to the left of nipple line, and there was a diffuse undulating impulse felt over the præcordial area. The heart's action was quick and very irregular in force and rhythm; no murmurs and no doubling of the sounds. There was slight roughening of the first sound about the apex, apparently of exocardial origin, as it became quite inaudible as the ear was

removed from the stethoscope. The case was then looked upon as one of dilated hypertrophy, with numerous pericardial adhesions. She improved very much, and was soon able to move about without much inconvenience, and on March 22 was sent to the Convalescent Institution, from which she returned on April 1 in even a worse condition than on her first reception. Cardiac dyspnœa great, and troubled with constant sickness. Countenance anxious and depressed.

On inspection of the chest there was seen an undulating impulse over the greater part of the præcordial area, which was found to have the following rhythm on attaching two small levers over the most prominent points of visible impulse, namely, one over the apex beat in the sixth interspace about one inch to left of nipple line, and the other at the upper and left side of the xyphoid cartilage. The lever over the apex has a distinctly double undulation—the first outward and upward movement being the most distinct. The movement of the lever over the right ventricle intervenes between the two undulations of that over the apex. On palpation, the apex impulse occurs first; then that over the right ventricle; and thirdly, a weak second pulse at the apex. The two periods between the summits of these three impulses are of about equal duration, while the pause that intervenes between the last element and the first of a succeeding cycle is equal to or rather more than the other two intervals. On auscultation there are heard three distinct sounds over the ventricles, the first being loudest around the apex, the second over the right ventricle, and the third at the apex. The second sound is very feeble at the base, and not audible over the ventricles, while at the aortic cartilage it is duplex. The pulse is slow, and exhibits the following rhythm with great regularity,—namely, there is a moderately strong beat, followed by a weak, scarcely perceptible beat; and then, after a considerable pause, there is a strong beat again, thus :—



April 10. The patient presents an anxious, depressed countenance, with pale, rather shrunken, cheeks. There is considerable orthopnœa, but no lividity. Conjunctivæ bile-tinged.

On examination of the heart to-day its action is found to have considerably varied since last note. There is a general undulating impulse visible over the ventricular area, which, on attaching three levers to the surface, is found to have the following rhythm. The first lever is placed at the left side of the xyphoid; the second in the fifth interspace, half an inch within nipple line; and the third in the sixth interspace, one inch beyond nipple line. The two outer levers are seen to move at the same time, while the inner alternates with the other two. This is corroborated by palpation, with the following slight refinement. The impulse in the sixth interspace precedes that in the fifth interspace, by which it is quickly followed, while that at the xyphoid alternates with the impulse in the sixth

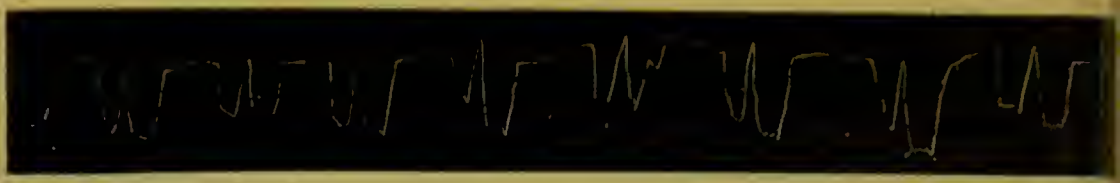
interspace. Owing to the general heaving, it is difficult to say which point is to be regarded as the apex beat, but the lowest and most external point of distinct pulsation is in the sixth interspace one inch beyond nipple line, and percussion also locates the apex in this situation. While it is noted that the most pronounced periods of pulsation, or what may be called the acme of impulse, over the left and right ventricles alternate, yet the pulsations run into one another. At the xyphoid cartilage there is a distinct retraction synchronous with the beat in the sixth interspace.

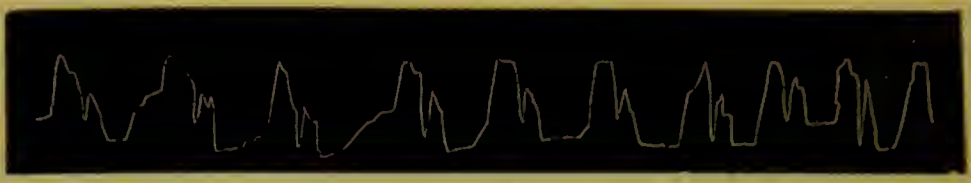
On auscultation, at the apex, there is a soft blowing short murmur, not audibly connected with any sound, but corresponding with the impulse in this situation. This is followed in a short interval by a sharp, clear sound; and this by two sounds which follow one another in quick succession. In the fifth interspace the murmur is still audible, though not so intense in character, and here there is a sound associated with it, which the murmur succeeds. The other sounds are here distinctly heard, but somewhat modified, not being so intense, and the last two present the cantering character of a duplex second sound. At the xyphoid cartilage there is a sharp, clear ringing sound, followed by a duplex second sound. When one bell of the differential stethoscope is placed over the impulse in the sixth interspace, and the other over the xyphoid cartilage, the murmur is first heard, and then a sharp, clear sound which corresponds with the first sound over the right ventricle. The other two sounds are a double second sound, the last of the two being most intense over the xyphoid, and the last but one over the apex. When one bell is placed over the aortic and the other over the pulmonic cartilage, the second sound is distinctly double. The duplication is also heard over each area, though not so intensely as when both areas are auscultated at the same time. The primary element of the double sound is loudest at the aorta, and the second element most intense at the pulmonic cartilage.

The superficial area of cardiac dullness measures 4½ inches, and deep area 5½ inches transversely, vertical line 3½ inches. The hepatic dullness is large, the lower border being within an inch of the level of the umbilicus, measures 5½ inches in line of sternum, 5 in line of nipple, and 5 inches in line of axilla. Pulse 38 per minute, very weak, but quite regular.

The cardiographic tracings reveal the fact that at each site of pulsation the impulse was double, consisting of two beats, or almost two ventricular systoles The period between the two beats was too short to be discernible by touch; all the finger could estimate was the duration of the beat and the period at which it attained its acme. As the finger detected that the height of pulsation over the left ventricle alternated with that over the right, so the tracings show that the first pulsation is most pronounced over the left ventricle and the second over the right ventricle. The first pulsation is the product of the left, and the second of the right ventricle, but although each pulsation is almost complete in itself, and presents the usual characters of a complete cardiac impulse, yet they do no represent a complete systole of their respective ventricles, for in that instance the two first could not precede the two second sounds. There is almost but not

complete ventricular asynchronism; the commencement of the right ventricular systole, with closure and tension of the tricuspid valve, occurs before the completion of the left ventricular systole, with consequent recoil of the aorta and tension of its semilunar valves, The impulse of the left ventricle





Taken at the apex in the sixth interspace, one inch to the left of nipple line.



Taken in the fifth interspace, one inch within nipple line.



Taken in the fifth interspace, two inches within nipple line.



Taken in the fourth interspace, midway between nipple and mesial iine.



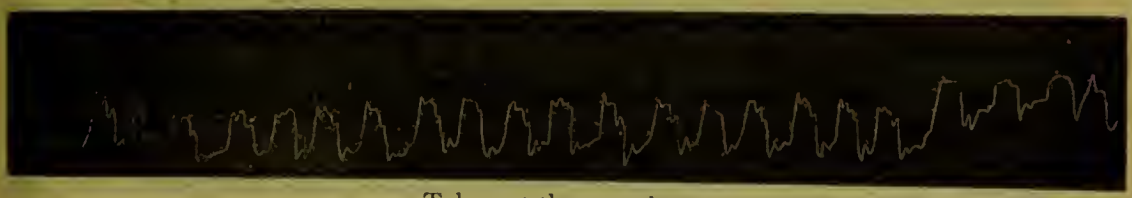
Taken in the fourth interspace, near the sternum.

is most pronounced at the apex and also in fifth interspace, but it is also communicated through the medium of the right ventricle to the rest of the cardiac area; on the other hand, the impulse of the right ventricle is strongest over its surface, but it is also communicated through the left

ventricle to that part of the parieties over the superficial portion (fifth and sixth interspaces) of the latter. Some of the cardiac cycles present three pulsations, thus showing a return to the previous condition where there were two systoles of the left ventricle for an intermediate one of the right.

April 17. Great orthopnœa, hard troublesome cough, with frothy expectoration. Face pale and rather sallow. Conjunctivæ slightly bile-tinged. Extremities cold. Pulse 90; short, weak, and irregular in force and rhythm. Occasionally troubled with vomiting of greenish biliary matter. Appetite poor. Urine high-coloured; no albumen.

Physical examination.—There is a general undulating impulse over the præcordial area, but the asynchronous action of the ventricles is not so marked as that last noted, owing to the more rapid action of the heart. The pulmonic second sound is accentuated, while the aortic sound is weak. When one bell of the differential stethoscope is placed over the aortic and the other over the pulmonic cartilage, the second sound is distinctly double, the last element being the loudest. At the apex beat in the sixth interspace beyond nipple line, there is a systolic murmur followed by a clear sound, and then a duplex second sound. As the stethoscope is moved towards the sternum, the murmur becomes gradually fainter, and at the xyphoid cartilage is almost lost; and here there is a clear ringing sound, followed by a sharp double sound. When one bell of the stethoscope is placed over the left and the other over the right ventricle, the cantering action of the heart becomes very marked, the whole four sounds being distinctly audible. On proceeding to take some cardiographic tracings, the heart's action became much excited, owing to a violent seizure of coughing. The beats ran up to 120 per minute (about one-half of which were not felt at the wrist), but the peculiar cantering action, especially the double second sound, was still appreciable to the ear, though the asynchronism is not recorded in the tracing.



Taken at the apex beat.

April 25, Sunday. I received a message to visit this patient, as she had been much worse since yesterday. I found her very anxious and distressed, with severe stabbing pain in right sight, breathing hurried, catching, short, and shallow. Skin hot and dry; tongue furred; appetite bad; bowels regular; troubled with frequent vomiting of mucus. On examination of chest I found pleuritic friction at base of right lung, but there was then no evidence of fluid effusion. I ordered poultices, a diaphoretic mixture, and hypodermic injections of morphia.

I was unable to see her on the 26th, and on the 27th I found that she had died suddenly about an hour before my arrival. The house surgeon did not notice

much alteration in her condition, and when he last saw her, a few minutes before her demise, he did not observe any marked change, or approach of the quickly fatal issue.

Post mortem.—I. W., æt. 31, died suddenly at 12.15 P.M. on April 27, 1880. Autopsy at 4 P.M. on April 28. Post-mortem rigidity well marked, lividity of dependent parts. Body spare, but not much emaciation. Subcutaneous fat scanty, except in abdominal parietes. On opening the abdomen, the liver is seen to be much enlarged and depressed, the lower margin reaching below the umbilicus. Below the liver, a small portion of the stomach and small intestine, with omentum, which is almost devoid of fat, are visible.

On opening the chest there is found a large quantity of serous fluid in right pleura, collected in a cavity which is bounded by the diaphragm, heart, root lower lobe and base of right lung. The right lower lobe is partially collapsed, and pressed upwards and outwards against the chest wall. There is a considerable amount of lymph flakes floating in the fluid. There is a thick layer of recent lymph coated over the right diaphragmatic pleura, the costal pleura as high as the fourth right rib, over the whole base and over the lower part of the surface of the right lung, over the inner surface of the cardiac pleura, and at the base of right lung. In the left pleura there are several ounces of clear serous fluid, but no lymph, nor any evidence of inflammation.

On laying open the pericardium there are found numerous adhesions over the whole anterior surface of the right ventricle and lower portion of the right auricle, each of about one quarter inch long, binding the two pericardial surfaces together. The left side of the heart is considerably enlarged, and a large triangular portion of it approaches the surface, having its base at the apex of about two inches wide. Over the front of the left ventricle, extending from the apex towards the septum, there is a vascular tuft of organised lymph, somewhat resembling the choroid plexus, of about an inch and a half in diameter. There are no adhesions binding the pericardial surfaces over the left ventricle and auricle. There are two or three ounces of serous fluid in pericardium. The left auricle is seen to be much enlarged and distended with fluid blood.

On removing the lungs, the left is found to be a little congested, but otherwise healthy. Right lung : the anterior surface of middle and lower lobes, at the root of lung, the inner and lower surfaces of lower lobe, the lower third of the posterior surface of lower lobe, and the pleural surfaces between the three lobes, are thickly coated with a layer of recent lymph. The rest of pleural surface of right lung is free from lymph. The middle lobe and anterior half of lower lobe are quite collapsed, and sink in water. There is slight emphysema at apex and along the anterior margin of the upper lobe. The rest of the lung is congested, but otherwise healthy.

Heart weighs 23 ounces, the enlargement being chiefly noticeable in the left side, the ventricle being hypertrophied and the auricle much dilated ; the right auricle is also large, though much less than the left. On tying the pulmonary artery and superior vena cava, and then filling the right ventricle

and auricle through the inferior vena cava, they are found to hold 70 fluid drachms of water. Filled in a similar manner, the right ventricle is found to hold 17½ fluid drachms. On tying the aorta and two of the pulmonary veins, and then filling the left ventricle and auricle through the other two veins, they are found to hold 91 fluid drachms of water, of which 20 fluid drachms are contained in the left ventricle.

Thus, the right auricle holds 52½ fluid drachms.
,, ,, ventricle ,, 17½ ,, ,,
,, left auricle ,, 71 ,, ,,
,, ,, ventricle ,, 20 ,, ,,

The cavities of the heart were now laid open, and the muscular structure was found quite healthy and free from any fatty degeneration. The right auricle is large, and its walls thin, but muscular structure healthy. The tricuspid orifice admits three fingers easily, and the valves are healthy. The right ventricle is normal in size, with rather thickened walls and large columnæ carneæ ; the walls measure 3 to 4 millimetres as apex, 5 millimetres in the body, and 4 millimetres at the base. The pulmonery valves are quite healthy, as also the pulmonary artery.

The left auricle is very large and its walls much attenuated, not measuring more than 1 millimetre in complete thickness, including the epithelial layer, muscular structure, and visceral pericardium. The cavity of the left ventricle is perhaps slightly larger than normal, and its walls much thickened, and the *musculi papillares* are very much hypertrophied. The walls measure 7 millimetres at apex, 10 millimetres about one inch from apex, 14 millimetres in centre of body, and 11 to 12 millimetres at base. The structure is of a red colonr, and free from any fatty striation. The mitral orifice is contracted, so as just to admit the thumb. The mitral cusps are so united as to constitute an infundibulum, and present an oval opening formed by their hard, thickened edges. This ring or button-hole opening is quite hard and cartilaginous to the feel The valves are much thickened, but quite smooth on both their auricular and ventricular surfaces, except a very slight coating or roughening, with fibrinous specks along the edges of the opening. The thickened and slightly roughened edges prevent very accurate apposition, but meet very closely and are in good condition for developing sound. The aortic valves are quite healthy : there are a few small atheromatous patches above the aortic valves, but aorta otherwise smooth and healthy.

Large nutmeg liver, weighing 73 ounces. Spleen normal. The kidneys weigh 16 ounces, congested, otherwise healthy.

A very interesting case of complete "Asynchronism of the two sides of the Heart" has been recorded by Dr. Bindley,[1] and illustrated by cardiographic tracings. The following extract

[1] *Lancet*, July 9, 1881.

from his notes will be sufficient to describe the nature of the case :—

"Over the apex there was a double beat, the first element of which had its point of maximum intensity at the outer margin of area of impulse, and seemed to come up to the chest wall from a short distance within. Almost immediately the sensation of this beat had ended the second element began, and appeared to start out of and from the spot where the first was best felt, and slide on, upwards and inwards, beneath the finger, at the same time communicating a distinct stroke—in fact, more distinct than that of the first element. At a spot an inch inside the left mammary line in the fifth space it was possible, with the end of one finger, to perceive the characteristics of both beats. If the end of the finger were shifted a little outwards, the first of the two strokes became more evident; if shifted a little inwards, the second was more prominently felt, while the force of the first was diminished. The two beats were followed by a pause of longer duration than the time between the beginnings of the two beats. The pulsation in the carotid was synchronous with the first element; with the second there was no trace of a carotid pulse."

In the second and third left intercostal spaces, about three-quarters of an inch from the edge of the sternum, over the conus arteriosus, there were pulsations synchronous with the apex beat. Pulse, 62 in the minute.

Starting with the sound resulting from the closure of the mitral valve, "the following sequence and rhythm were determinate, the irregularity recurring in a regular manner :—

1....21′....2′.....1....21′....2.....1..., &c., &c.

The figures represent the first and second sounds respectively, and the dotted lines indicate the relative length of the intervals between the sounds. The figure 1, synchronous with the carotid impulse, signifies the mitral closure; 2, the aortic; 1′, the tricuspid (which followed immediately upon the aortic); 2′, the pulmonary. None of the sounds were loud, but were of a perfectly pure character. There was no murmur. The relation of the sounds to the double beat was equally plain, 1 corresponding to the first beat, and 1′ to the second. The basic beat in the second space was synchronous with 1′."

Another case of demonstrable asynchronism I observed in an old lady aged 74, with rather rigid arteries, but who, with the exception of a little flatulence, was in as good health as might be expected at her age. Her heart was perhaps large,

but not larger than is usually found in persons of advanced years. The apex beat was situated in fifth interspace in nipple line, and was rather diffuse and heaving, which was more apparent on account of the body being very spare of flesh. The first sound was duplex, and the doubling was best heard in the fourth interspace, but the first element was most intense at the apex, and the second towards the sternum over the right ventricle. The duplication was distinct at the apex, and here the first element was strictly synchronous with the impulse. The second sound was single, but accentuated at the aortic cartilage. When the fingers were pushed well up into the epigastrium, the pulsation of the right ventricle could be distinctly felt, and this was perceptibly later than that at the apex.

Some time ago I had under my care an old lady, aged 58, suffering from mitral regurgitation, with dilated hypertrophy of the right ventricle and rigid arteries. Her general symptoms were those resulting from impeded pulmonic circulation. In this case there was no constant and regular duplication of the cardiac sounds, but there was frequently doubling for a few cardiac revolutions, which was accompanied by irregular cardiac contraction or jumping of the heart, and therefore not very readily appreciated.

On Nov. 5, 1880, the day previous to her death, the following conditions of the heart were observed :—The pulse was 30 per minute, regular. The impulse was situated in fifth interspace, about one inch beyond nipple line, and consisted of a distinct double beat, the first part being most pronounced. Cardiac action regular but slow.

On auscultation, both sounds were found to be double over the greater part of the præcordium. The doubling of the first sound was best heard about the fifth interspace, between nipple and sternum. As you approached the apex the first element became associated with the mitral systolic murmur, and at the apex was almost entirely replaced by this murmur. The following were the conditions at the apex :—A short mitral systolic murmur coincided with the first beat of a double impulse. This was followed by a sharp, clear ringing sound, which corresponded with the second beat, and these were quickly succeeded by two short, dull sounds. At the base, and at the aortic and pulmonic cartilages, the double second sound, especially with the aid of the double stethoscope, was heard to be well marked, the pulmonic element being the last and loudest. There was a greater interval between the two first sounds than between the two second, so that the second first sound was as nearly associafed with the first second sound as with the first first.

Some might feel Inclined to consider this an instance of two systoles to one pulse, one of the systoles being insufficient to raise the semilunar valves, or at least to propagate the wave to the periphery. If this were the case, the abortive systole must have been the second, because the second impulse was the weakest, and, as a matter of fact, the pulse was associated with the first beat. This supposition is readily disposed of on the following grounds : the first beat and sound were attended by a murmur, but not so the second ; and moreover the character of the sounds was quite different, the second element being very clear

and ringing. Again, the second sound was double, and both elements followed the two first sounds. The case is only explicable on the theory of partial asynchronous ventricular contraction.

Austin Flint, in a very interesting article[1] on this subject, follows Bouillaud, the original observer of doubling, in ascribing reduplication to the "failure of the two ventricles to contract in unison. According to this explanation, the tricupsid and mitral valves are made tense, not simultaneously but successively, in consequence of the contraction of one ventricle being completed before that of the other; and the semilunar valves of the aortic and pulmonic artery expand alternately, instead of coincidently, since, in consequence of difference in time between the completions of the right and left ventricle, the recoil of the coats of each of these two vessels does not occur at the same instant." While Dr. Austin Flint believes in the asynchronous theory of reduplication, yet he fails to enter into the mechanism of asynchronism.

Dr. George Balfour believes in the asynchronous origin of reduplication, but adopts Potain's erroneous explanation of its mechanism. He says that in normal reduplication "the first sound becomes reduplicate because excess of blood pressure in the heart retards the closure of the tricuspid valve, and the second sound is reduplicated because excess of pressure in the aorta accelerates the occlusion of the aortic valves."[2] Such an explanation is not merely inconsistent but at entire variance with the influence which the respiratory process plays in carrying on the circulation of the blood. I would rather say that the excess of blood in the right heart during deep expiration stimulates the right ventricle to commence contraction first, and consequently the tricuspid valves are tensely closed before the mitral, thus giving rise to a double first sound; but, owing to the overloading of the ventricle and the increased obstruction in front, or want of freedom in the flow through the pulmonary circuit, the right ventricle is longer in emytying itself; and hence, although it had the start of the left, it has not completed its

[1] *Diseases of the Heart*, second edition, 1870, p. 322.

[2] *Diseases of the Heart and Aorta*, 1876, *p.* 34.

contraction before it, so there is no doubling of the second sound under these circumstances.

Regarding doubling of the second sound, I have clearly shown[1] that in inspiration, the tension in the aorta is not "increased," but is diminished. The primal elements of physiological doubling of both sounds are associated with the right side of the heart, and not, as Dr. Balfour asserts, with the left side.

Dr. Balfour says:—"Reduplication of the second sound is of frequent occurrence in mitral constriction, due to the great pulmonary congestion, always present in these cases, which so increases the tension in the pulmonary artery as usually to accentuate, but sometimes so to accelerate the closure of its valve as to cause the pulmonary second sound to anticipate that of the aorta, and thus to reduplicate the sound. You can see that when reduplication takes place, closure of the valves must occur before the ventricular systole is finished; they are shut, therefore, in the face of a still advancing current, and in such circumstances, as you can readily suppose, forcible closure is not always possible, and accentuation is frequently absent."[2]

This is certainly a very "forcible" explanation of a common fact, without a tittle of evidence to support it. It does not seem to have occurred to Dr. Balfour that when the increased obstruction in front thus prolongs the right ventricular systole, then the closure of the aortic semilunar valves may anticipate that of the pulmonic. The first element in these cases being aortic, does not require such a remarkable explanation to account for its non-accentuation.

Dr. George A. Gibson, in a very interesting paper on "The Sequence and Duration of the Cardiac Movements,"[3] gives some cardiographic tracings, taken from a case of presternal fissure, which show asynchronous recoil of the aorta and pulmonary artery. In this case there was never any audible doubling, hence he concludes that "reduplication of the recoil

[1] *Medical Times and Gazette*, Jan. 1877.

[2] *Diseases of the Heart and Aorta*, 1876, p. 33.

[3] *Journal of Anatomy and Physiology*, Jan. 1880, p. 234.

may be present without reduplication of the sound being audible."

He gives the following reasons for this view:—"According to Despritz, quoted by Carpenter, the minimum number of complete vibrations required to produce an appreciable musical sound is eight per second, and Savart, also quoted by Carpenter, fixes the minimum at from seven to eight per second. Contradictory statements are made with regard to the duration of sonorous impressions on the ear, this being said by some observers to be longer and by others shorter than the duration of ocular impressions. Foster says that the 'ticks of a pendulum beating 100 in a second are readily audible as distinct sounds.' In the reduplication on the tracing the arterial recoils are separated by the tenth part of a second; yet it must be borne in mind that the sound accompanying each is not a simple noise, but, caused as it is by the vibrations of a membrane and a column of fluid with the secondary vibrations of the surrounding structures, approaches the character of a musical note. It seems much more probable that the one sound is continued into the other than that the reduplication should play at hide-and-seek with the observer."

Dr. Hayden, while he believes that doubling of the second sound is due to asynchronous closure and tension of the two sets of semilunar valves, advocates another theory for doubling of the first sound. He says, "The *first sound* of the heart coincides with the impulse and the first two-thirds of ventricular systole; it is the product of a twofold cause, namely the impulse of the ventricles and the sudden tension of the auriculo-ventricular valves and chordæ tendineæ . . . , and it seems to be a compound of a 'thud' and a 'click'; the former, as I believe, caused by the impulse of the ventricles, and the latter by the sudden tension of the auriculo-ventricular valves and chordæ tendineæ. . . . Reduplication of the first sound is the result of its resolution into the above mentioned two elements."[1]

From this explanation I must dissent, as the impulse begins

[1] *Diseases of the Heart and Aorta*, 1875, pp. 115, 116.

with the muscular rigidity, which gives rise to the intra-ventricular pressure, and this in turn to valve closure and tension, with the resultant "click"; it is evident that under no circumstances can the "click" *precede* the "thud." Again, as the impulse continues throughout, at least two-thirds of the ventricular systole, and certainly beyond the period of greatest intra-ventricular pressure, it is plain that the "click" cannot *succeed* the "thud." No doubt the first sound is frequently recognisable to the ear as a compound, but this resolution into its component elements of impulse and valve tension can never be looked upon as a reduplication. In short, the impulse includes the closure of the auriculo-ventricular valves, and, consequently, the "thud" includes the "click." Moreover, doubling is generally better heard over the body of the ventricles where the sound of impulsion is usually eliminated, than at the apex where it is most pronounced.

A somewhat similar, though in several respects different, view was held by Hope. He says: — "The valvular click gives smartness and intensity to the commencement of the first sound, and in feeble hearts, in which the sound of extension and of *bruit musculaire* are absent, the click alone is heard, causing the first sound to be identical in quality with the second. This occurs, for instance, in dilatation with attenuation. The sound of muscular extension superadds bluntness and loudness to the valvular click, and is probably a principal cause of the extraordinary intensity of the first sound, often observed in violent palpitation. It differs from the sound of costal percussion with metallic cliquetis, which imparts a double character to the first sound." [1]

"The cause of the *metallic cliquetis* is simply this: — The heart in gliding forwards and upwards during its systole, strikes with its apex against the inferior margin of the fifth rib, and thus creates an accidental sound, attended with cliquetis when the blow is smart. It may be prevented at pleasure by pressing the edge of the stethoscope or anything else into the intercostal space, by which that space is put, internally, on the same plane

[1] *Diseases of the Heart*, 1839, p. 63.

as the rib, over which the heart then glides without catching. I have never found the sound to occur in any but the meagre; because, in the well-conditioned, the intercostal spaces are full and resistant, and, consequently, the edge of the rib is not exposed. It is not necessary to dwell here on this phenomenon, but I may remark that I have for many years noticed the first sound to be *double* in some patients. The cause is, that the blow of the heart against the edge of the rib is a little later than the first sound."[1]

This theory, though very ingenious, is too fanciful to merit any serious consideration. Doubling of the first sound, though often fugacious, is not so easily extinguished as Hope would have us believe. In the meagre, skin and bone are good conductors of sound, and doubling is easily detected; whereas in the well-conditioned, fat is a bad conductor, and the intensity of even the normal first sound, and the area over which it is audible, are greatly lessened.

Hayden believes, the first element of a double first sound, to be the sound of impulsion, or, as Hope calls it, the sound of *muscular extension*, and the second element to be the valvular click. Hope, on the other hand, believes the first to be the valvular click, and the second to be due to an accidental smart blow against the inferior margin of the fifth rib, and not to the ordinary sound of *muscular extension*.

Bellingham [2] observed this so-called *metallic cliquetis*, and adopted Hope's explanation.

The late Dr. Sibson[3] fully recognised the clinical importance and frequent occurrence of reduplication of one or both sounds in cases of Bright's disease, and he ascribed the doubling to asynchronism in the action of the two sides of the heart and the great vessels attached, but his explanation of the mechanism and causes of asynchronism is very meagre, and in many respects unsatisfactory. His explanation, that doubling of the first sound in cases of Bright's disease is due to a more tardy and prolonged

1 *Diseases of the Heart*, 1839, p. 41.

2 *Diseases of the Heart*, 1853, pp. 109, 110.

3 *Lancet*, April 11, 1874; *British Medical Journal*, Jan. 13, 1877.

contraction of the left than the right ventricle is perhaps an extension of his view, that the normal first sound "begins and continues with a rumble, and ends with an accent or sharp sound, *which is coincident with the extreme contraction of the cavity*" (the italics are mine).[1] Dr. George Johnson very pertinently asks, if that be the case, why then is the second sound not invariably reduplicated in these cases; or if the absence of that phenomenon can be accounted for by the much greater strain on the aorta than the pulmonary artery, enabling the former to overtake the latter in producing tension of its valves, then we should expect that, normally, the high tension of the aorta would produce premature closure of its valves, with consequent reduplication of the second sound.

In cases of double second sound, Dr. Sibson also associates (in this instance correctly) the final element with the artery in which there is high tension, which leads to prolonged ventricular contraction and consequently delayed arterial recoil. In carrying his argument out to its natural conclusion, it would seem that a duplex second sound occurs in extreme cases of asynchronism, when even the greater strain of the aorta cannot enable it to overtake the recoil of the pulmonary artery. We might, again, ask, why then is a double second sound not invariably preceded by a double first.

It is not the high tension *per se* which causes the reduplication, but it is the effect which it produces in the capacities and irritability of the ventricles, and its interference with the uniform distribution of the blood. That ventricle which is relatively the best supplied with blood, and which best retains its muscular irritability, initiates contraction, first raises its intra-ventricular pressure to its height, and tensely closes its auriculo-ventricular valve, thus giving rise to the first element of a double first sound. It does necessarily follow that the ventricle which begins will first end contraction, for the length of the period of systole varies. The shorter the systole, the greater and more active the power, relatively to the mass to be moved and to the obstacle in front.

[1] *Medical Anatomy*, 1869.

Professor D'Espine, of Geneva, in an interesting communication on clinical cardiography, delivered at the International Medical Congress, London, 1881 (to which I then had an opportunity of replying), and afterwards published in an extended form in the *Revue de Médecine* Paris, 1881, advances some ideas on reduplication, to which I now wish to refer.

"III. The physiological tracings of the ventricular contraction, as well as the shock tracings, show that the systole is not a single muscular contraction, but a compound of successive efforts amongst which may be distinguished two principal pulsations—(*a*) the mitral pulsation of the ventricle, which results in forcing the blood of the heart into the aortic reservoir; (*b*) the arterial pulsation, which drives the blood of the aorta into the ultimate limits of the arterial tree. These two pulsations are marked in the carotid pulse; the second alone exists normally in peripheral pulses, such as the radial or femoral."[1]

These statements are not supported by his tracings. The so-called mitral pulsation is certainly represented in the radial pulse by the percussion wave; and in his simultaneous tracings the apex of the radial is only slightly behind the first summit in the shock tracings, and certainly before the so-called "arterial pulsation," which is represented by the final summit in the apical impulse and the tidal wave in the arterial pulse.

"IV. Mitral pulsation is sometimes single, sometimes double. A first single sound corresponds always to a simple mitral pulsation; on the contrary, the more the two pulsations are manifested and distant the clearer will be the reduplication of the first sound, and the more like the true *bruit de galop*."

"In the dog and in man, mitral pulsation is single in the normal state. In the horse it is double, and corresponds to a double click of the auriculo-ventricular valves, very distinct from the presystole. The presystole counts for nothing in the production of the *bruit de galop*."

"There is never a reduplication of the first sound, because the two ventricles are perfectly synchronous in the first portion of the systole. There may be, on the contrary, reduplication of the second sound,

[1] International Medical Congress, 1881, *Abstracts of Papers*, p. 126.

because the closure of the sigmoids may be asynchronous in the aorta and pulmonary artery."

My auscultation of horses is rather limited, but so far as I have gone I have found the first sound single. It is unnecessary for me here to repeat my views on doubling and the mechanism of asynchronism; but I think I have shown that asynchronism at the commencement of ventricular systole is not an uncommon condition.

What is a *bruit de galop* but rapid reduplication, and, in my opinion, undoubtedly due to asynchronous valve tension, though Professor D'Espine explains it by an imaginary double click of the left or right auriculo-ventricular valve. It is quite impossible that a double click could occur without an intervening ventricular relaxation, which would be equivalent to abortive systoles. Dr. D'Espine says that "the true mechanism of the double valvular click remains to be discovered." I would rather say that the double valvular click itself remains to be discovered, and is likely to remain so, as it is non-existent; but the mechanism of doubling is an established fact. With regard to his explanation of tracings [1] from cases of "acute interstitial nephritis—true galop (néphrite interstitielle aiguë — vrai galop)." I believe that what he calls the beginning of ventricular systole is really the commencement of the auricular systole. If he be correct, and if the drum of his cardiogram moved at the rate of "four centimetres in a second," then in the first portion of the figure, the radial pulse is delayed two-ninths of a second, and in the second portion the carotid pulse is delayed one-eighth of a second, which is scarcely credible. Even Professor D'Espine previously tells us that "the error which arises from the time which separates the aortic from the carotid pulse may be ignored; it is at the most from 2 to 3-100ths of a second," and we can scarcely believe that it took a vigorous ventricle the remainder of the time to raise the semilunar valves.

I should now like to refer very fully to the exceedingly able,

[1] Fig. 2. *International Medical Congress Abstracts*, p. 128. Fig. 9. *International Medical Congress Transactions*, vol. ii. p. 150. Fig. 21. *Revue de Médecine*, Paris 1881, p. 30.

clear, and unprejudiced writings of Dr. Sansom on reduplication of the heart sounds; but as this article has already assumed rather lengthy proportions, I can only treat of the points of difference between us. I have, however, much pleasure in referring the reader to his excellent manual on *Diagnosis of Diseases of the Heart*, which is perhaps the best little book on the subject in the English language; to the *Proceedings of the London Medical Society*, 1880; and to the *Medical Times and Gazette*, 1881.

Dr. Sansom[1] "finds it difficult to associate reduplicate first sound in any causal way with differences of intra-ventricular pressure, seeing that long-persisting variations in such pressure and disturbance of balance must be so common, whilst the phenomenon itself is so rare." But I never said that difference in pressure, or what might be better expressed as mere difference in the quantity of blood in the two ventricles, was capable of giving rise to duplication. It is difference in the balance of pressure and in the irritability of the ventricles which gives rise to doubling of the first sound, for instance, to speak in very general terms, if one ventricle require two ounces of blood and the other four ounces, to stimulate them to contraction, and if each can receive its respective quantity in the same given time, then there may be perfect synchronism at the beginning of systole; moreover, the larger ventricle may only half empty itself during contraction, and consequently it is half filled at the beginning of diastole. Doubling of the first sound occurs when the balance of blood-supply enables one ventricle to receive its quantum of blood sufficient to overcome the inhibitory action of the vagus, and initiate contraction in advance of the other.

Normally, I believe, that both sounds are well heard over the whole præcordium, though I know that some place a more narrow limit on their different areas of audibility. In some conditions, both intrinsic and extrinsic to the heart, the sounds are very much intensified, while in others they may be almost inaudible, or each sound may be heard only over a very limited area. In duplication, the different elements may be heard over the whole præcordium,

[1] *Diagnosis of Diseases of the Heart*, 3rd edition, London, Churchill, 1881, p. 115.

or their different areas of audibility may be so limited that it requires the double stethoscope to appreciate the fact that there is doubling. This is quite in harmony with the views which I have expressed. If you believe that the first sound of the heart is in great part composed of the sound resulting from the tension of the tricuspid and mitral valves, then when there is a resolution of the causes of sound, there will naturally be a diminution in intensity, and consequently a lessening in the areas of diffusion.

Dr. George Johnson [1] objects to the asynchronous explanation of double first sound, on the grounds that the doubling is perhaps more frequently heard over the body of the heart, than at the apex, where the first sound is most intense. But it must be remembered, that in addition to the valvular element, the other causes of the first sound, viz., the muscular sound, and the sound of impulsion or muscular extension, are most pronounced at the apex, and gives it its prolonged and booming character; while, when you pass inwards over the septum, you get partly rid of these two latter causes of sound, and retain only the valvular element; besides you come more within the area of the tricupsid sound, and owing to the right ventricle overlapping and lying in front of the left, the mitral sound is more readily propagated to the tricuspid area than the tricuspid sound is to the apex.

Dr. Johnson says that, when duplication of the first sound is heard over the body of the ventricles, but is absent at the apex, "the part of the double sound which is lost as we pass from base to apex is always the first or presystolic part." But I have here related three cases where the first element actually coincided with the apex beat, and I could give other cases if necessary. Besides, the apex beat or impulse is not always formed by the left ventricle, but just by that part of the cardiac globe which, during contraction, comes into closest proximity with the chest walls. When you have got a hypertrophied and dilated right ventricle due to emphysema of the lungs without any corresponding affection of the left ventricle, the impulse is almost invariably caused by the former.

[1] *Lancet*, May 13, 1876; *British Medical Journal*, April 28, 1877; and *Medical Times and Gazette*, Feb. 10 and March 3, 1877.

Drs. Sansom and Guttmann argue against the asynchronous valvular theory of the mechanism of a double second sound in cases of mitral stenosis, because they have frequently found the doubling more marked over the ventricles than at the pulmonic and aortic cartilages. But, is not the normal second sound also very frequently better heard over the ventricles? I recently examined 100 prisoners, taken indiscriminately, and in fully one-half the number, I found that the second sound was much louder over the ventricles than either at the aortic or pulmonic cartilages. The aortic element may be loudest at the aortic cartilage, and the pulmonic at the pulmonic cartilage, but yet duplication may not be marked at the base, because these elements are not readily conveyed to the different arterial sites. The pulmonic element is not easily conducted to the aortic cartilage, but readily to the right ventricle, and the same may be said of the aortic element. Besides the conduction of the different elements to the opposite arterial sites depends on the tension of the arteries at the time, *e.g.*, from my standpoint, supposing the pulmonic element in a given case, to be the primal one, then the left ventricle not having ceased contraction, the aorta is still tense and in good condition for conducting the pulmonic element to the aortic cartilage; but suppose the aortic element to be the primal one, then by the time the tension of the pulmonic valves occurs, the aorta is comparatively lax, and not in good condition for conducting the pulmonic element to the aortic cartilage. Similar reasoning might be applied to the conduction of the aortic element to the pulmonic cartilage.

Guttmann says,[1] that duplication of the second sound is, "absent in the more marked cases of mitral contraction, precisely the cases in which the conditions most favourable to the postponement of the closure of the pulmonary valve are present in the highest degree." But, as I before stated, there may be failure in the muscular force of the right ventricle, it may act as a blood reservoir, and may only partially empty itself during systole, and so its partial systole may end as soon as the complete systole of the left ventricle; and these are just the cases in which such

[1] *Handbook of Physical Diagnosis, New Sydenham Soc. Trans.*, 1879.

failure is most apt to occur. Here then is a prognostic point of some value. If, in a case of mitral stenosis, you have got doubling of the second sound which during the progress of the case is abolished, and if this abolition be accompanied with signs of dilatation of the right ventricle, you have a grave element introduced which may be averted for a time by rest, digitalis, &c., but which augurs badly.

The slower the heart's action the more easily is duplication appreciated, and very rapid action of the heart is sufficient to abolish it. This is readily explained by the asynchronous ventricular theory, but not so by any auricular theory, because the asynchronism between the auricular and ventricular contractions is so marked, that mere rapidity of action should not abolish it. Besides, frequently all you have got to do to develop a latent auricular systolic murmur is to make the patient run a few times round the room.

This leads me up to the auricular theories.

Dr. Johnson holds [1] "that *the contraction of a dilated, and especially of an hypertrophied auricle, becomes sonorous, and that the first division of the double first sound is the result of the auricular systole.*"

This theory is based on the analogy of the auricular systolic murmur, and the auricular pericardial friction. The causes of these two phenomena are patent enough, so it became necessary for Dr. Johnson not merely to assert that the first element was the result of the auricular systole, but to explain how this auricular systole gave rise to a first sound, and accordingly he stated that "there are three influences which, either separately or combined, may cause an audible sound during auricular systole — first, the sudden muscular tension of the walls of the auricle; secondly, the impulse against the chest walls; and, thirdly, the forcible impact of blood driven onwards by the auricle against the stationary blood within the ventricle." Drs. Williams and Broadbent convinced Dr. Johnson that this last element could have no influence in the

[1] *Lancet*, May 13, 1876; *British Medical Journal*, April 28, 1877; and *Medical Times and Gazette*, Feb. 10 and March 3, 1877.

causation of the first sound, and I wish I could convince him that the first two have equally nothing to do with duplication.

Dr. Johnson plainly first held that an hypertrophied auricle was a chief cause of duplication, but he evidently recognised the fact that duplication occurs where there is no hypertrophy, and so he added dilatation. To those who might feel inclined to adopt his theory, but who are fond of generalisation, it might possibly seem a pity that he had troubled himself about the state of the auricle, because, with such limitations as those, how could he explain physiological duplication? When the auricle is dilated the contraction is very feeble; so how, then, could you have an audible muscular sound? and where there is feeble contraction there cannot be much impulse against the chest walls. Besides, only a very small portion of the left auricle appears on the anterior surface, and yet Dr. Johnson believes that in one half or more of his cases the first element of sound is due to the left auricle. In mitral stenosis again the auricular systolic murmur ceases when there is much dilatation. In one of my cases the auricle was so thin that I don't believe that it contracted at all, but simply acted as a blood sinus or reservoir. Moreover, the first element coincided with the ventricular impulse. I have lately heard duplication of first sound in two cases of Bright's disease in an early stage, where there was no dilatation of the left ventricle, and where the first element coincided with the impulse.

Dr. Sansom's theory "that in some cases the energetic systole of the auricle suddenly floating up the curtains of the mitral valve may so put them on the stretch as to cause a sound—the click of valve tension," seems very plausible at first sight, so far as duplication of the first sound is concerned. It might seem reasonable to suppose that at the end of diastole the auricular systole might fill the ventricle so full as to cause tension of the valves; but when you come to analyse the sequence of events more closely, you will find that the auricular systole is not the immediate cause of floating up the valves, because as long as blood is rushing through the mitral orifice, it keeps the valves open, and it is only when the blood ceases to flow that the recoil of blood within the ventricle can float up and make the valve tense. Again, when the auricle

has filled the ventricle, the ventricular systole begins, and so continues the valvular tension, which the recoil of blood within initiated, and thus we would just get a prolonged sound, not a double one.

Potain noticed a form of seeming reduplication of the first sound, which he termed *bruit de galop* in cases of hypertrophy of the heart associated with granular kidneys. He considered that the first element of the apparent duplication was presystolic. No doubt this *bruit de galop* is nothing but rapid duplication, though my equestrian friends will agree with me that it is not a galop but a canter. As to his believing that the first element is presystolic, that is merely asserting that the first occurs before the second, but it is no proof whatever that the first is auricular systolic in origin.

Guttmann has observed such "presystolic" sound in a number of cases of cardiac hypertrophy. He considers "that the auriculo-ventricular valves are to a certain extent rendered tense at the end of diastole—that is, in the presystole, but this tension is normally so feeble that no sound results; but if the walls of one of the auricles undergo hypertrophy as the consequence of some valvular lesion, the corresponding auriculo-ventricular valve is put more sharply and thoroughly on the stretch by the contraction of the auricle, and in this way the conditions necessary to the production of a presystolic sound are realised." No doubt the auriculo-ventricular valves are closed immediately at the end of auricular systole by what I have termed the recoil of blood within the ventricle; but then the ordinary tension of the auriculo-ventricular valves occurs at the commencement of ventricular systole, and what I might term the acme of intra-ventricular pressure, which occasions the sharp tension of the auriculo-ventricular valves, and occurs immediately before the opening of the semi-lunar valves, is so quickly attained that there can be no appreciable interval between what we might term the recoil tension, and the tension due to the ventricular contraction.

Guttmann says that in cases of mitral stenosis "the reduplication of the second sound originates at the narrowed mitral orifice itself, as it so often vanishes completely on increasing

the force of the heart's action, and gives place to a loud diastolic murmur; there is therefore nothing forced in the inference that the two elements of the phenomenon in question, which form a sound which is always more or less muffled or impure, are in reality the component parts of a murmur.—It has also been conjectured, on the other hand, that the first part of the divided sound is simply the diastolic pulmonary sound, and that the second is produced, towards the end of the diastole, by the contraction of the hypertrophied left auricle,—a theory which would yield a plausible explanation of those cases in which the reduplication remains, even when the diastolic murmur is developed."

Even supposing that the auricular contraction can give rise to a first sound or element of first sound, it is impossible to conceive that the auricular systole ever occurs so early in diastole as to be the cause of the second element of a duplex second sound, but for the sake of argument let us suppose that it can, then how can it cause sound by suddenly flapping up and putting the valves on tension, while they are kept open by blood rushing in to an only partially filled and dilating cavity—conditions which render their valves and chordæ tendineæ lax?

Again, granting that in mitral stenosis the valves are rigid and united together, so that they form a kind of funnel, and cannot lie back against the walls of the dilating ventricle, and thus blood can easily get behind them and float them up. But then the chordæ tendineæ and musculi papillares would be quite lax, and would allow considerable play to the valves before they would be put on tension. Besides, if the ventricle were dilating rapidly so as to keep the chordæ tendineæ slightly stretched, and allow the blood to apply a certain amount of tension to the valves, then the blood, rushing through a narrow orifice to a larger space beyond, would give rise to a *fluide veine* murmur and not a sound. Any such rapid dilatation of the ventricle so as to be a powerful sucker would imply a great waste of power, which is contrary to all the laws of nature, and would be resisted by the atmospheric pressure. Dilatation is a comparatively gradual process, like slowly raising the piston of a syringe.

In mitral stenosis, the final element of a duplex second sound is always the loudest, and in my opinion is undoubtedly pulmonic in origin, and is due to the prolonged contraction of the right ventricle, with consequent delay in the recoil of the pulmonary artery. Owing to the high tension in the artery, the sound is more intense and abrupt than the primal one which arises in the aorta. It never resembles a murmur.

Dr. Sansom has recognised the improbability of the auricular systole occurring so early in the diastolic period as to give rise to the second element, and accordingly he fortifies his theory by stating that the "reaction of the distended pulmonary veins and left auricle may be sufficient to cause tension—so it may occur previously to the auricular contraction, the latter occasioning or reinforcing the presystolic murmur, separated by a slight interval from the second element of duplication."

The veins are not composed of strong elastic fibre, like the aorta and pulmonary artery. What, then, is this "powerful reaction"? It is simply the reaction of hyperdistension with feeble contraction, which is a very simple matter indeed.

The great length which this paper has now assumed prevents me considering the bearing of doubling on diagnosis, prognosis, and treatment. I must therefore forego dealing with this part of the subject at present. The recognition of a duplex sound is usually a matter of no great difficulty, but the mere observation of a fact stands for very little if you be not capable of interpreting that fact, and fully estimating other facts of which it is but the effect. The successful practitioner is he who does not consider the most minute and recondite fact unworthy of his observation and investigation, and who at the same time clearly weighs the interdependence of one factor on another, and takes a broad and comprehensive view of the whole individual case.